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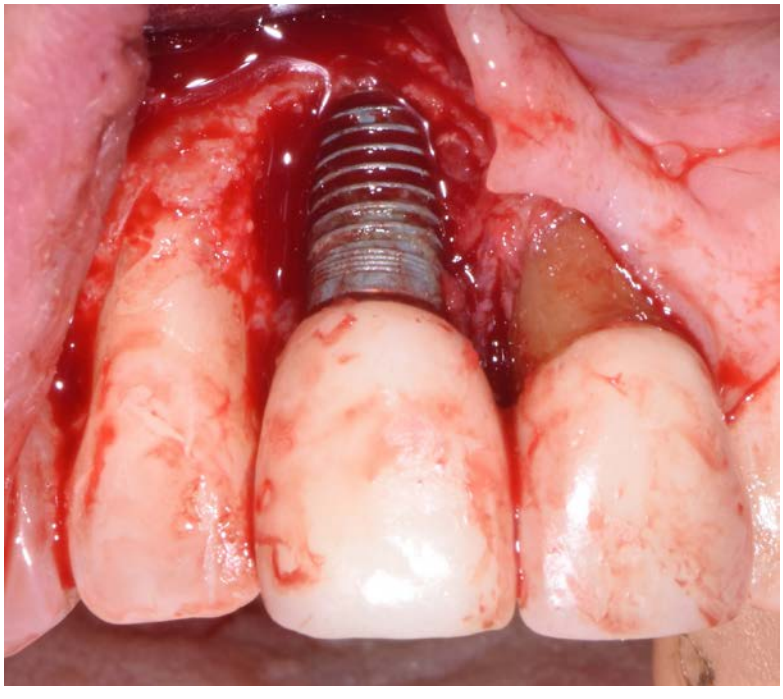

All screwed up: Strategic considerations for failing implants

A PEER-REVIEWED ARTICLE | by Adam Saltz, DMD, MS, MPH

ABSTRACT

Failing implants can result in major esthetic and functional changes. These implants, however, should not be immediately condemned to removal. An understanding of biology, risk assessment, and treatment planning will help in the decision-making process when assessing diseased implants. Different maintenance strategies, regenerative materials, and surgical techniques have increased implant survival rates at costs significantly less than dental implant replacement. It is worth revisiting ways to manage peri-implantitis and peri-implant complications to ensure an implant is appropriately maintained, treated, or removed.

EDUCATIONAL OBJECTIVES

Upon completion of this course, the dental professional should be able to:

1. Diagnose peri-implant conditions using the latest classification systems.
2. Describe the etiologies for peri-implant disease and identify strategies to address these etiologic factors.
3. Discuss local and patient-level risk factors that lead to implant failure.
4. Utilize evidence-based treatment strategies for failing dental implants, employing appropriate nonsurgical and surgical protocols to maintain, treat, or remove ailing/failing implants.

Dental implants can fail, and fast. Treating peri-implant disease and its related complications can result in major esthetic and functional changes. An understanding of biology, risk assessment, and treatment planning will help in the decision-making process for ailing/failing implants. Different nonsurgical and surgical techniques, regenerative materials, and home-care regimens offer new opportunities for implant maintenance. Learning how to diagnose and manage peri-implant disease via a targeted, anti-infective approach can improve outcomes and more appropriately inform a decision to treat, maintain, or remove a diseased implant. Strategies for removal and replacement of hopeless implants will also be discussed to minimize risk factors that led to their initial failure.

Diagnosing peri-implant conditions

There are fundamental differences between teeth and implants. In the

healthy periodontium, the periodontal ligament and supracrestal tissue attachment stabilize the tooth[1]; whereas the peri-implant mucosa lacks such structures and the soft and hard tissue attachment associated with the implant differ considerably from natural teeth.[2] Apical to the peri-implant epithelium, two connective tissue compartments surround the implant body.[2] The more superficial layer is predominated by dense collagen and is acellular in nature, resembling scar tissue, while a provisional tissue matrix, abundant in fibroblasts, directly contacts the implant surface. As the mucosa matures over the following two to three months, collagen fibers run parallel from the alveolar bone to the implant fixture.[2] This loosely arranged orientation exposes the implant to bacterial or mechanical insult, in addition to the weaker, long junctional epithelial attachment.[2,3] Immune surveillance cells, such as neutrophils, remain present throughout.[3]

The keratinized mucosa then extends to the oral mucosa around most implant sites.[2,3] It is highly collagenous and outlined by squamous orthokeratinized epithelium.[2] This band is generally narrower around implants than the contralateral teeth. Its importance in implant stability and disease treatment, however, remains controversial because available short-term studies are more focused on its association with inflammation and marginal bone loss than the effects of peri-implant phenotypes on implant survival rates.[2,3] Implant surface characteristics and prosthetic components affect the quality and maturity of the peri-implant tissue attachment.[3]

These soft tissue compartments are vulnerable to disease progression and should be closely monitored during healing and loading. Sites should be visualized, probed, and palpated during recall visits to detect changes in attachment, inflammation, and bleeding tendency. Initial measurements should be taken at baseline loading for future comparison. It is worth noting that a lighter probing force around 0.20 N should be used around implants to prevent overdiagnosis as bleeding on probing (BOP) is readily elicited with heavier probing forces, even on healthy implants.[4]

BOP is often the first sign of peri-implant mucositis.[5] This is an inflammatory condition in the adjacent mucosa with no supporting bone loss. It is reversible to a point. Its conversion to peri-implantitis is difficult to predict but accelerated by certain risk factors, such as cigarette smoking, poorly controlled type 2 diabetes mellitus, and a lack of oral hygiene.[5] In fact, a lack of maintenance led to peri-implantitis in 43% of cases over a five-year period.[6]

Peri-implantitis represents the progression of this lesion into the alveolar bone. When comparing human biopsy material, these lesions are much larger than periodontitis samples and extend past the pocket epithelium with increased plasma cells, neutrophils, and macrophages. They are associated with an accelerated, nonlinear pattern of bone loss because peri-lesional cells have greater mRNA levels that code for highly active and destructive cytokines.[7] Clinically, peri-implantitis is identified by pocketing of more than 6 mm with bleeding and/or suppuration on probing. These tendencies were the best diagnostic indicators of progressive bone loss over a nine-year period.[8] In the absence of previous radiographs, bone levels of more than 3 mm apical to the implant shoulder are diagnostic of peri-implantitis.[3,5]

The above criteria are outlined by the 2017 American Academy of Periodontology/European Federation of Periodontology World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions (AAP).[3,5] The prevalence of peri-implantitis has increased as people outlive their teeth and seek implant prostheses to replace them. In fact, Sweden's Dental and Pharmaceutical Benefits Agency found that the number of implants placed has doubled in their population over the last decade with about two implants per individual.[7] The prevalence of peri-implantitis is widely variable because of different case definitions for clinical and radiographic parameters and studies limited to academic settings with unrealistic controls. The accepted occurrence of peri-implantitis is around 40% in a random population sample with severe forms close to 10%, findings similar to periodontitis.[7]

This information has also shifted current thinking of implant "success." Implants had long been considered successful if they lacked mobility, progressing vertical bone loss, and/or did not violate local or regional anatomy leading to pain or paresthesia, regardless of their esthetic or functional appearance.[9] Today, implants are considered successful when they present with the above criteria and pink, firm peri-implant tissues that do not bleed are truly stable.[10] By taking a more comprehensive approach to implant stability that includes hard and soft tissue parameters, clinicians can more aptly detect marginal changes before they lead to catastrophic failure.

Etiologies for peri-implant disease

There are three main drivers of peri-implant disease. Bacteria normally live among mixed species in unique spatial arrangements, known as biofilm, that adhere to teeth and implants.[11] Titanium forms a thin, biocompatible dioxide layer upon placement.[2,7] This changes the electrostatic forces and

ionic bonding properties of the local biofilm, which can accelerate bacterial adhesion and colonization, and thereby shift the microbiome toward dysbiosis. The resulting biofilm represents an overgrowth of periopathogenic bacteria normally present in smaller amounts.[7,11]

Technologies using different hybridization and polymerase chain reaction (PCR) techniques have found that peri-implantitis lesions have mixed, highly variable, and mostly gram-negative anaerobes.[7,11] More recently, gram-positive *Staphylococcus epidermis* has been isolated from certain lesions and could be responsible for more advanced infections. As with periodontitis, the biofilms also differ between shallow and deep pockets, with increasing virulence more apically.[11] Such biofilms are often nonresponsive to beta-lactam antibiotics, leading to unwanted failures and costs.[11]

Metallosis complements this destructive sequalae. This phenomenon was discovered from histologic evidence on failing prosthetic joints in the orthopedic literature.[12] It describes a pathogenic process that is initiated by virulent, opportunistic microbes and exacerbated by localized titanium particle and ion release into the neighboring tissues. The titanium dioxide layer cannot be repaired once it is damaged and exposed to the oral cavity. Implant placement, mechanical debridement, frictional wear, micromovement, and a combination of these factors called tribocorrosion contribute to metallosis.[12] This has dramatically moved implant disease treatment away from multistep, multiproduct protocols toward minimally traumatic but complete biofilm removal.[7]

Lastly, there has also been a recent shift in what defines osseointegration. Implant insertion directly affects alveolar bone development and local bone homeostasis.[13] Osseointegration is now perceived as a continuous and dynamic host-defense reaction known as the foreign body equilibrium.[13] This could explain why immune cells are always present in the peri-implant tissues.[3] If the soft tissue inflammatory front approaches the marginal bone in response to sustained bacterial or mechanical injury, osteoclastic activity is induced. This results in bone loss, fibrosis, macrophage activity, and systemic pro-inflammatory cytokine release.[13] Implant treatment and maintenance should therefore reflect and focus on controlling these etiologies to help minimize damage to the peri-implant complex.

Local and patient-related risk factors

It is important to understand the local and systemic risk factors that affect implant survival and how they contribute to disease progression. Certain conditions are more impactful. For example, heavy cigarette smoking (i.e., >15 cigarettes daily) has been significantly associated with peri-implantitis after six to seven years.[14] Nicotine and its metabolites induce a shift toward more anaerobic bacteria via decreased oxygen perfusion and a suppressed immune response, which leads to oral dysbiosis.[11] A recent meta-analysis found a near threefold risk in developing peri-implantitis among smokers, especially when using more than 20 cigarettes daily.[15]

Tobacco cessation should therefore be encouraged as part of implant therapy. Smoking fewer than 10 cigarettes per day can reduce implant disease risk and offer a more realistic first step in reducing cigarette consumption among heavy smokers.[7,15] It was previously thought that former smokers would eventually return to a risk profile of never smokers; however, new evidence suggests that each year after quitting there is only a 3.9% reduction in risk of periodontal/peri-implant attachment loss with a significantly decreased risk after 21 years.[16,17]

A similar effect is noted with systemic diseases, such as diabetes mellitus and osteoporosis. Patients with hyperglycemia or an HbA1c above 5.4% had significantly higher bleeding scores and peri-implant bone loss compared to healthy controls.[18] Diabetes mellitus alters the diversity of the gut and oral microbiomes, leading to dysbiosis through altered calcium metabolism.[18] The inflammatory burden associated with dysglycemia and hyperglycemia leads to an accumulation of advanced glycation end products and reactive oxygen species that impairs cell signaling and collagen turnover.[18] No significant differences in clinical outcomes were found among those with osteopenia or osteoporosis taking antiresorptive drugs, as long as their comorbidities and oral hygiene were well controlled.[19] Patients with complex medical histories should consult their physicians prior to dental surgery. A combination of the above risk factors may accelerate peri-implant complications and failure in a susceptible host.

After thorough review of a patient's health history, an intraoral exam should be completed. The severity and type of periodontitis can dramatically influence implant health over time. In a prospective study, the 10-year implant survival rate was 90% for those treated for severe periodontitis. However, 47.2% of these patients required antibiotic or surgical intervention as part of their maintenance.[20] This cohort was then followed for another 10 years. There was a 14.6-fold risk of implant loss when patients were not maintained.[21] Treated implants had a survival rate of 67% with a majority requiring further surgery, or worse,

implant removal.[22]

Patients with a history of periodontitis without supportive care are particularly vulnerable to peri-implantitis because of other systemic comorbidities. Bacterial plaque is the strongest risk indicator for peri-implantitis.[7] Efforts should be made to improve daily oral hygiene and maintenance compliance with active therapy pursued on a need basis.[23] Patients should be made aware of their disease risk and possible adjunctive procedures after placement as part of the initial treatment discussion to set the appropriate expectations and reinforce the importance of proper instrumentation and home care.

The type of periodontitis may also convey additional risk. Previously "aggressive" or "juvenile" periodontitis patients—whom we would now categorize as Grade C periodontitis—have a 14-fold greater risk for peri-implantitis.[24] Further, those individuals with generalized distributions of more rapidly progressive periodontitis are four times more likely to experience implant failure.[25] Altogether, dental implants should be placed in patients with well-controlled periodontal and systemic conditions and established maintenance regimens. This is, in many ways, more important than the implant surgery itself.

Decision-making in implant disease treatment

It should be clear that peri-implantitis stands as its own disease entity and thereby requires a highly specialized and targetic approach. Historically, ailing implants were treated exactly like their tooth counterparts with resective surgery.[7] For example, in a randomized clinical trial, osseous surgery was performed around diseased implants with or without removal of the exposed implant threads. There was significantly decreased pocketing and attachment levels after three years in the implantoplasty group.[26] However, this does not model current thinking. Implantoplasty has been associated with greater titanium dissolution and violates the titanium dioxide layer, initiating dysbiosis and metallosis—the perfect storm for propagating an ongoing foreign body reaction.[13,27] This respective procedure is better coupled with hard and/or soft tissue augmentation to reduce surface convexities for improved coverage and regeneration. The rationale to eliminate these threads for better plaque control is significantly reduced with today's hand and power instruments that will be reviewed as part of implant maintenance.

Regenerative strategies became popular with their long-term success around previously diseased teeth. They include the use of bone replacement grafts, membranes, biologic factors, or combinations thereof.[7] Specialized flap designs, using a single-flap or papilla sparing technique, further increase vascularity to these augmented sites.[7] Froum et al. described an eight-step decontamination protocol using sodium bicarbonate, saline, tetracycline, 0.12% chlorhexidine gluconate, and multiple bioactive agents prior to connective tissue grafting or resorbable collagen membrane placement.[28] However, 20% of treated implants required one or two more procedures after 10 years to stabilize disease.[28] Justifying the time, expense, and healing requirements to only possibly or temporarily save an ailing implant should be heavily considered when managing peri-implantitis.

Recent clinical trials and systematic reviews that compared different reconstructive methods to conventional flap surgery found negligible differences in probing depth reduction and bleeding scores between groups.[7,29,30] Radiographic bone gain was consistently reported but is subjective and therefore difficult to interpret repeatedly and fairly. The shape of the intrabony defect seems to have an inconclusive effect on treatment outcomes.[7] While soft tissue recession improved with regenerative therapy, no magnitude of clinical effect was observed, especially after one year.[7,30] The role of the keratinized mucosa around implants is still debated and controversial in its influence on reconstructive surgery.[3,7]

It is now recognized that most nonsurgical and surgical workflows are harmful to the implant body and immune cell response.[12,13] Ultrasonic scalers can release titanium ions into the adjacent mucosa and damage implant surfaces.[29] Surface modifiers, like polyprotic acids and local antibiotics, applied to exposed implant threads cause discoloration, pitting, and corrosion, driving metallosis.[12,30] The adjunctive use of different systemic antibiotics in nonsurgical and surgical therapy led to generally favorable pocket reduction and marginal bone stability up to one year but had an overall low quality of evidence.[7,11,31] Despite its rich history in the periodontal literature, 0.12% chlorhexidine gluconate altered the viability of wound healing cells, like osteoblasts and fibroblasts, with significantly greater cytotoxicity and apoptosis.[30] At this time, gauze soaked in normal saline or a saline solution are considered safe and effective methods for implant surface decontamination.[30]

Other conservative options have quickly gained attention. A nonsurgical step is first advised to remove plaque and reinforce proper home care.[31] Air-polishing with erythritol powders can effectively remove implant-bound biofilm, calculus, and debris without surface damage because they are small, highly irregular

particles.[30,32] The use of lasers, photodynamic therapy, videoscopes, and other noninvasive technologies requires further investigation, where most are limited to animal studies or short-term findings.[33,34] In fact, according to the 2023 Clinical Practice Guidelines of the European Federation of Periodontology, lasers were not recommended as part of nonsurgical treatment of peri-implantitis nor surgical implant surface decontamination because of this paucity of evidence.[31]

Unfortunately, despite the clinician's best efforts, implants still fail. Their survival rates decrease with each reattempt. A recent systematic review reported a mean survival rate of 86.3% up to five years after implant re-treatment with a significant drop in these rates at 68.7% among smooth-surfaced implants.[35] Other studies reported implant survival rates of second and third reattempts as low as 71%.[36] Underlying patient-level risk factors, implant surface topography, surgical site anatomy, residual bacteria at failed endodontic sites, and traumatogenic occlusion may affect re-treatment success rates.[35,36] A history of periodontitis and bone grafting at time of initial implant placement also negatively impacted the five-year survival rate of replaced implants in a retrospective study.[7,36]

The decision to remove an ailing implant should therefore consider relevant risk factors, treatment interests, and finances. Implant removal can result in major hard and soft tissue defects, requiring repeated corrective surgery, or worse, esthetic failures. In clinical practice, a hopeless implant may be retained

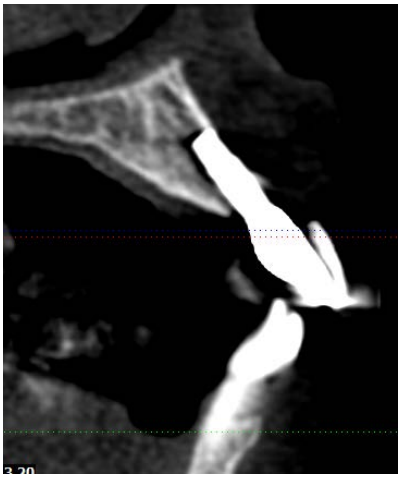

FIGURE 1. CBCT imaging noting the buccal fenestration at implant no. 8

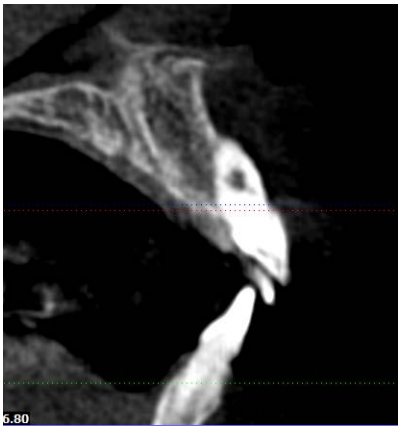

FIGURE 2. CBCT imaging of nonrestorable no. 9

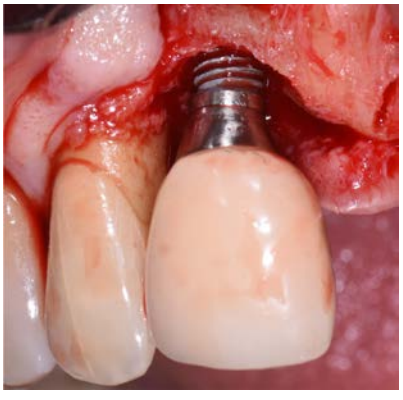

FIGURE 3. Full thickness flaps reflected to visualize implant and root surfaces and bone

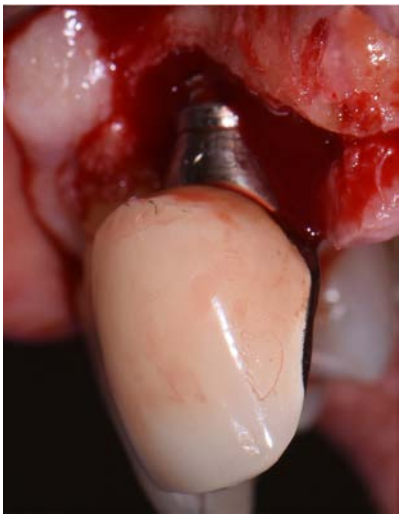

FIGURE 4. Intraoperative lateral view of the overcontoured crown no. 8

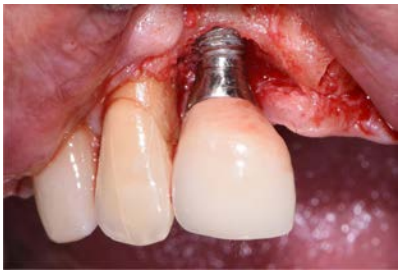

FIGURE 5. Interproximal osseous recontouring and implant surface debridement performed to eliminate the shallow defect to improve hygiene and restorative access

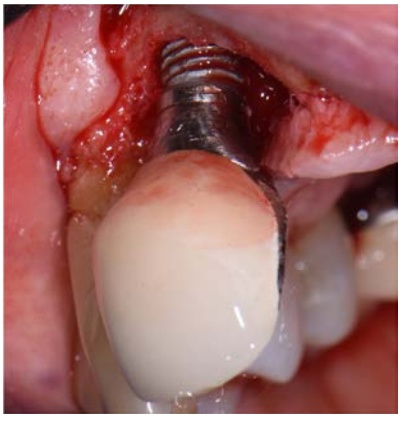

FIGURE 6. Intraoperative lateral view of the modified crown no. 8 with the appropriate subgingival emergence

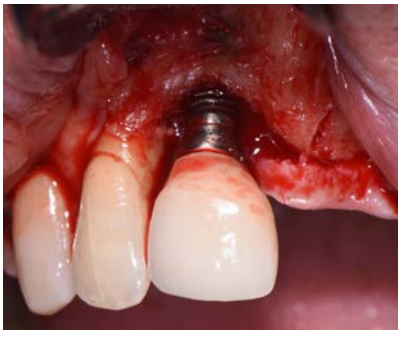

FIGURE 7. Buccal contour grafting performed to eliminate the ridge concavity along nos. 8 and 9 and prepare site no. 9 for future implant replacement

with strict maintenance and good home care until it becomes symptomatic and/or the patient can afford removal and site reconstruction for possible replacement.[7,31,37] Some implants, however, are so compromised through attachment loss or implant malpositioning that the resulting prosthetic complications, osseous breakdown, and recession require an extensive interdisciplinary approach to manage or replace, potentially impacting the adjacent teeth or implant(s). The time, expense, and recovery to re-treat these failures is not always worthwhile. Care should be taken to address risk factors as part of the initial implant planning to create realistic patient expectations and ensure long-term implant health.

Implant maintenance and home-care regimens

Implant maintenance should reflect a patient's dental and medical needs. For example, periodontal patients, who are already more susceptible to peri-implantitis, should continue three- or four-month recalls.[31] At these appointments, dentists should inspect the implant and prosthesis for tissue changes, signs of infection, or occlusal overload and intervene accordingly. Hygienists should use the aforementioned hand and power instruments and workflows to safely and effectively remove supra- and subgingival plaque, calculus, and cement around teeth and implants.[31] Radiographs should be taken once annually unless more frequent imaging is otherwise needed.[37,38]

Oral hygiene measures now consider current models of peri-implant disease. Submucosal floss has been associated with peri-implantitis, as remnants were increasingly noted around defective implant-abutment connections and exposed threads.[38] Water flossers have consistently demonstrated greater plaque removal and better tissue response than floss and interproximal brushes around implants, which was confirmed in a recent randomized-controlled clinical trial.[39] Patients with full-arch, removable implant appliances are encouraged to clean their implant fixtures and prostheses at least twice daily. Complete fixed restorations should be removed, disinfected extraorally, and reinserted with new screws on a regular basis determined by the patient's plaque control and other risk factors.[40]

Case report

A 73-year-old Caucasian male presented to a private practice in South Portland, Maine, interested in options to address peri-implantitis on tooth no. 8 and nonrestorable no. 9 (figures 1-9). The patient had a medical history significant for osteoarthritis. He smoked at least 10 cigarettes daily for 40 years. At the time of his initial exam, 6 mm pocketing with heavy bleeding and suppuration and horizontal bone loss were noted at implant no. 8. These measurements were underestimated given the overcontoured, ridge-lapped crown that limited probing access, especially on the buccal aspect. This was placed and restored five years ago by another surgeon. Tooth no. 9 was deemed nonrestorable from expanding resorptive and carious lesions.

The following surgical plan provides a treatment modality for each risk factor or problem noted in the initial evaluation. Different materials and techniques were reviewed, and consents were obtained. His treatment plan included:

- Tobacco cessation
- Implant flap debridement and osseous recontouring of 8
- Extraction of 9 for future implant placement with a surgical guide using a digital workflow
- Site preservation of 9 with contour bone grafting along 8 and 9
- Refabrication of implant crown 8

On the day of surgery, sulcular incisions were made from teeth nos. 7–10 for full-thickness flap elevation and alveolar crest visualization. Tooth no. 9 was first extracted in a minimally traumatic fashion. The malpositioning of implant no. 8 presented a restorative concern and contributed to the hard tissue deficiency in the right anterior maxilla. Implant no. 8 was thoroughly debrided using titanium hand scalers of equal hardness and gauze soaked in normal saline. The implant crown was reduced at the cervical convexity to improve hygiene and restorative access. After socket degranulation and implant debridement, a 3 mm fenestration and 3-4 mm dehiscence were noted at the facial plate of sites 8 and 9, respectively. Despite ample palatal and apical bone around 9, immediacy was not considered because of these tissue defects and active disease present.

Periosteal releasing incisions were then prepared for improved flap passivity. The facial plate was augmented with an allogeneic mixture of 80% mineralized/20% demineralized ground cortical granules using a lateral onlay technique and resorbable collagen membrane. Flaps were reapproximated with monofilament sutures for primary, tension-free closure. The patient was prescribed an antibiotic regimen, short course of steroids, and stabilized chlorine dioxide rinse during the perioperative period. These agents minimize systemic infection, inflammation, and damage to the implant body and wound-healing cells.

The restorative dentist delivered a maxillary Essix retainer to replace missing no. 9 to avoid lateral dispersion of the grafted materials. The patient was made aware of the unfavorable prognosis of implant no. 8 and

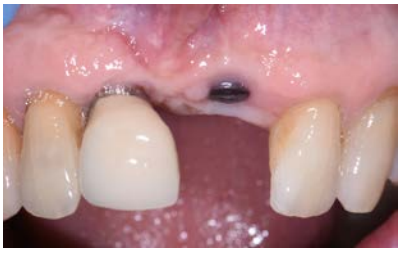

FIGURE 8: Six weeks after implant placement #9.

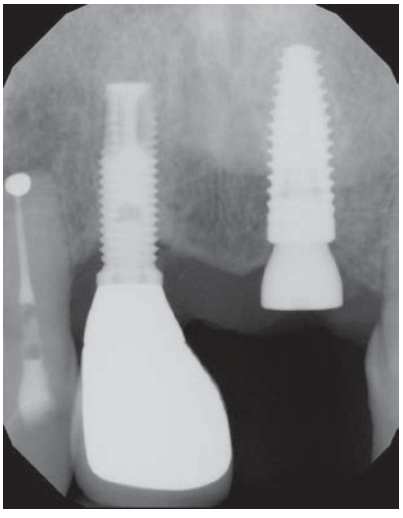

FIGURE 9: Prerestorative radiograph confirming normal healing of the treated sites.

understands it may be lost in the future, even with treatment rendered. He ultimately had limited finances, hoping to restore 9 first and retain 8 as long as possible with the modified crown. He agreed to be seen more frequently for recare.

With the etiologies now stabilized, the patient was maintained every four months in preparation for implant no. 9. He was encouraged to use interproximal brushes until about three months of ridge healing, when he began use of a water flosser for subgingival irrigation. He quit smoking prior to surgery and efforts of sustained cessation were reinforced at each visit. The above treatment plan first controlled local and patient risk factors for failing implant no. 8 and the tissue deficiencies at no. 9, so implant no. 9 could safely be placed and maintained in a nondiseased site. Figure 8 shows six weeks after implant placement, and Figure 9 is the prerestorative radiograph confirming normal healing of the treated sites.

Conclusion

Risk factors that precipitate oral dysbiosis, metallosis, or the foreign body reaction and systemic conditions that alter host susceptibility drive the prevention and treatment of peri-implant disease today. Nonsurgical and surgical interventions should use instruments and materials compatible with the implant, abutment, and restoration. Maintenance visits and oral hygiene instructions should satisfy site- and patient-specific needs for long-term health and treatment success. While the etiologies for implant failure are exhaustive and ever changing, biologically and prosthetically driven treatment planning will minimize esthetic and functional complications. Taken together, the decision to retain, remove, and/or replace diseased implants should consider predisposing local and patient risk factors, treatment interests, and finances.

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1. In the healthy periodontium, the___ anchors the tooth in place.
   - A. Periodontal ligament
   - B. Cementum
   - C. Dentin
   - D. Circular fibers

2. The superficial connective tissue layer around a healing implant is mostly composed of:
   - A. Adipose tissue
   - B. Collagen
   - C. Blood vessels
   - D. Macrophages

3. Which of the following is not true regarding the keratinized mucosa around dental implants?
   - A. It contains squamous orthokeratinized epithelium.
   - B. It is highly collagenous.
   - C. It is generally wider than the contralateral teeth.
   - D. It is unaffected by the implant surface topography.

4. As part of each recall visit, implant sites should be:
   - A. Visualized
   - B. Probed
   - C. Palpated
   - D. All of the above

5. Initial probing measurements should be taken at ___ for future comparison.
   - A. Three months after implant placement
   - B. Six months after implant placement
   - C. Baseline loading
   - D. One-year postloading

6. What is the first notable sign of peri-implant mucositis?
   - A. Crestal bone loss
   - B. Bleeding on probing
   - C. Probing depths more than 4 mm
   - D. Keratinized, attached tissue width less than 2 mm

7. What is/are the risk factor(s) that can accelerate the conversion of peri-implant mucositis to peri-implantitis?
   - A. Cigarette smoking
   - B. Poorly controlled type 2 diabetes mellitus
   - C. Lack of oral hygiene
   - D. All of the above

8. What is/are the best diagnostic indicator(s) of implant disease around implants?
   - A. Pocketing more than 6 mm
   - B. Bleeding and/or suppuration on probing
   - C. Minimal attached tissue thickness
   - D. A and B only

9. Which of the following is not true regarding histologic lesions of peri-implantitis?
   - A. They are smaller than lesions seen at sites with periodontitis and similar clinical parameters.
   - B. They extend past the pocket epithelium.
   - C. They contain increased amounts of plasma cells and macrophages.
   - D. They have greater mRNA levels that code for pro-inflammatory cytokines.

10. In the absence of previous radiographs, bone levels more than ___ mm apical to the implant shoulder are diagnostic of peri-implantitis.
   A. 2 B. 3 C. 4 D. 6

11. According to Sweden's Dental and Pharmaceutical Benefits Agency, the average number of implants per adult in the Swedish population is:
   - A. One implant
   - B. Two implants
   - C. Three implants
   - D. Four or more implants

12. Which of the following pathogenic bacteria associated with peri-implantitis is gram-positive?
   - A. *P. gingivalis*
   - B. *T. forsythia*
   - C. *S. epidermis*
   - D. *T. denticola*

13. Which represents an overgrowth of periopathogenic bacteria normally present in smaller amounts?
   - A. Dysbiosis
   - B. Metallosis
   - C. Foreign body reaction
   - D. Biofilm

14. Which of the following is true regarding metallosis?

A. Titanium monoxide is highly susceptible to bacterial or mechanical insult.
B. The titanium dioxide layer cannot be repaired once it is damaged.
C. This phenomenon was first discovered in animal studies.
D. It is not related to or initiated by virulent, opportunistic microbes.

15. Which of the following occurs because of the foreign body reaction?

A. Fibrosis
B. Macrophage activity
C. Sustained cytokine release
D. All of the above

16. Smoking less than ___ can significantly reduce peri-implant disease risk.

A. 10 cigarettes daily
B. 15 cigarettes daily
C. 20 cigarettes daily
D. None of the above

17. Each year after quitting cigarette smoking, there is a ___ reduction in the risk of periodontal/peri-implant attachment loss.

A. 1.8% B. 3.9% C. 4.2% D. 6.6%

18. Patients with an HbA1c above ___ had significantly higher bleeding scores and peri-implant bone loss compared to healthy controls.

A. 5.4% B. 6.5% C. 7.0% D. 8.0%

19. What is the strongest risk factor for peri-implantitis?

A. Residual cement
B. Bacterial plaque
C. Type 2 diabetes mellitus
D. Obesity

20. The accepted occurrence of peri-implantitis is around ___ in a random population sample.

A. 20% B. 30% C. 40% D. 50%

21. Previously "aggressive" or "juvenile" periodontitis patients have a ___-fold greater risk for peri-implantitis.

A. 4 B. 8 C. 12 D. 14

22. Which of the following is true of nicotine and its metabolites and their effect on the periodontium?

A. They induce a shift toward more aerobic bacteria.
B. They decrease oxygen perfusion.
C. They exaggerate the host immune response.
D. They induce metallosis.

23. The criteria for implant "success" now includes:

A. No progressing vertical bone loss
B. Maintenance of regional anatomy
C. Lack of pain and related symptoms
D. Pink, firm tissues that do not bleed

24. Implantoplasty has the potential to initiate:

A. Dysbiosis
B. Metallosis
C. The foreign body reaction
D. All of the above

25. Which of the following agents alters the viability of wound-healing cells, such as osteoblasts?

A. 0.12% chlorhexidine gluconate
B. Ultrasonic scalers
C. Polyprotic acids
D. Local antibiotics

26. Which of the following agents causes discoloration, pitting, and corrosion?

A. 0.12% chlorhexidine gluconate
B. Normal saline
C. Polyprotic acids
D. Systemic antibiotics

27. What is a safe and effective method for implant surface decontamination?

A. Application of desiccant gels
B. Gauze soaked in normal saline
C. Thorough wash of polyprotic acids
D. Use of local antibiotics, such as tetracycline

28. Which clinical parameter seems to have a negligible effect on the success of regenerative therapy at dental implants?

A. Intrabony defect morphology
B. Soft tissue recession
C. Deep, baseline pocketing
D. A and B only

29. The mean implant survival rate is around ___ among smooth-surfaced implants.

A. 58.8% B. 68.7% C. 71.2% D. 86.3%

30. Which of the following factors negatively impacted the five-year survival rate of replaced implants in a five-year retrospective study?

A. History of periodontitis
B. Implant surface topography
C. Regional anatomy
D. Residual bacteria at failed endodontic sites

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